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The Diagnosis of Early Pulmonary Tuberculosis.

By C. D. PARFITT,
Calydor Sanatorium, Gravenhurst.

A prompt diagnosis of tuberculosis can be made probably in four out of five cases of the disease by the practitioner, simply by sound work. This is necessary especially for the elimination of suspected cases. Possibly not more than twenty per cent. of tuberculous patients will have a so-called "early" lesion, now known as "minimal," a term including duration, area and intensity of disease. An occasional error in over-diagnosis can be corrected and is safer for the patient than a diagnosis missed. It is important to hold the point of view that tuberculosis may be a possibility in any illness, even with other disease.

The essentials upon which a reasonable diagnosis may be founded are, an adequate history, a sufficiently detailed enquiry into present symptoms, a general physical examination with special attention in some few details to the lungs, observation in regard to temperature, pulse and weight, the prompt and repeated examination of the sputum, and possibly an X-ray examination. The safely applied tuberculin tests are mainly helpful for exclusion when negative on repetition; when positive they may prove a stimulus to further investigation. They should be used as a routine during infancy, childhood and adolescence. A bottle of approved old tuberculin, with scarifying needle for promptly doing a Pirquet skin test, and a sputum bottle in mailing case, for sputum, should always be on hand for suspected cases, in office or bag.

The diagnosis of the early cases must be one of elimination, and for this a history well taken and with an open mind is of first importance. Such a history, especially for the tuberculous, also helps prognosis. The history should, as a matter of routine, follow a good schema in order to avoid missing points. The patient's complaint should be noted, as given by himself. Molony found that entrance complaints drew attention to the chest in 86 per cent. of the Saskatchewan series. The present illness should be gone into in some detail, starting from the time the patient was last well, events being noted in chronological order, and symptoms elicited and classified as much as possible under the several body systems. Then a general survey of the personal history, past illnesses, and the functioning of the special systems in the past history, with the events in each group recorded in chonological order, is essential not only for diagnosis but for prognosis. Family history should include details not only relating to the immediate family, but also to grandparents, collaterals and consort. Other pulmonary diseases, possibly concealing phthisis, should be asked about. Enquiry as to possible contact infection, especially where children are concerned, should be made regarding servants and friends. Opportunities for the possible infection of adults will be noted in the personal history. There is good reason to believe that a third of adult patients have incurred reinfection after puberty.

Patients' statements regarding diagnosis of past, personal and family illnesses may be open to question and should be criticised. Every lead should be followed to a conclusion, so that some concrete opinion may be formed regarding an illness, actual or possible, at different stages of the history.

A detailed history is too frequently regarded as a boresome routine, but it is the key to most diagnoses which other methods only confirm. The considerable time involved in taking a good history is not so great for the practitioner, who judges facts immediately and notes those that are relevant, as for the student whose record is used by the physicians of the clinic, for whom all possible information is essential.

Respiratory infections, past or recent, and their possible complications deserve thorough enquiry, since they may conceal symptoms of onset. Symptoms relating to the gastro-intestinal tract, while sometimes due to abdominal tuberculosis, are in many cases due to the toxemia of the disease. Suggestive information may arise of glandular disease, with or without suppuration, bone or joint disease, a purulent otitis of insidious origin, and anal fistula, the result of ischio-rectal abscess. Irregular attacks of diarrhoea alternating with constipation may point to intestinal tuberculosis or tuberculous peritonitis, and the history of hematuria, perhaps with pyuria, to tuberculosis of the urinary tract. The genitalia require enquiry and often investigation. During pregnancy and lactation, enquiry about suggestive symptoms, and repeated examinations, would sometimes avert disaster. Patients with history of febrile attacks, recent indigestion, anaemia, asthenia and neurasthenia, all require investigation, since tuberculosis may be the cause of all such symptoms. Constitutional symptoms most significant of toxemia and which may also be characteristic of non-tuberculous focal infections elsewhere in the body are fever, slight or marked, night sweats, chills, acceleration of pulse, loss of weight, languor, lack of endurance, anorexia and indigestion.

Tuberculosis may be a concomitant of other disease, and while the patient is being treated for one disease he may be developing another. Indeed the primary disease may well have been the cause of breakdown of an already existing latent tuberculosis. Every diabetic should receive careful chest examination, and every tuberculous patient a careful urinalysis. Hoarseness or pain on swallowing demand a laryngeal examination. A cough persisting for a month, with or without sputum, requires a chest examination, and persistent sputum, even without cough, should receive repeated laboratory examination. A history of blood-spitting should, in this country, be considered tuberculous unless other explanation can be definitely found.

A general physical examination, including the nose, throat and larynx, is, of course, necessary. The sites of focal infection demand special investigation.

The examination of the chest should be thorough, but unprejudiced. Only a few points regarding it can be mentioned. The patient, when possible, should be examined in good light in the erect position, preferably on a stool. Note the type of chest and whether or not there is any scoliosis with consequent slight compensatory deformity, which will modify expansion and percussion and possibly respiratory sounds. Attention to this point may save wrong deductions in regard to possible underlying disease. Outlines marked on the skin greatly help to focus attention and make for accuracy. Mark the apex beat, the relative dulness of the heart, the margins of the lungs at rest, in the axilla and at the base posteriorly, and also the boundaries of the lower margins on inspiration and expiration. Then mark the projected isthmus of resonance at the apices, above the clavicle, and over the trapezius; note also the position of trachea. Many inferences are thus suggested at once.

Now note the quality of resonance, of percussion, breath sounds and vocal resonance, below the scapula on the pre-

sumed more normal side, where the characteristic tones for the individual are best obtained. These tones are standards for comparison. Using correct technique in percussion, allow normal discounts for modified notes and for skeletal peculiarities. Percussion in early cases sometimes yields very little trustworthy evidence, even for the experienced. Allow, also, reasonable discounts for breath sounds and voice sounds where normal modifications are known to exist. Learn somewhere the characteristics of faint pure bronchial breathing and be on the watch for its modified qualities. Auscultation of breath and whispering voice sounds should be done before any attempt is made to elicit rales. Surmise as to the pathological condition underlying the areas that show slight changes of respiratory sounds have been sadly upset by X-ray revelations, and judgment in regard to them must often be reserved. They suggest long-standing rather than recent disease.

Fine rales are today considered the earliest diagnostic signs of tuberculous infiltration; yet in patients admitted to the Trudeau Sanatorium, no rales were heard in more than half of those with minimal and in one-fourth of those with moderately advanced X-ray lesions. Rales may be heard during ordinary or deep breathing, but care must be taken to elicit rales not ordinarily heard during respiration. Expiration from the rest position followed by a single short slight cough, after which a moderate inspiration is taken, is the manoeuvre for the elicitation of such rales. In a tuberculous infiltration, sufficiently near the surface (within an inch and a half), rales may then be heard following cough and at the beginning of inspiration, not to be detected otherwise. The areas above the 3rd rib, in the high axillary regions, supra spinous fossae, interscapular regions, and at the angles of the scapulae, should be carefully auscultated for such rales. Persistent pulmonary rales in the upper half of the chest, probably, but not invariably, indicate tuberculosis.

Pleuritic rales are sometimes difficult to differentiate. They are usually unaffected by cough. Atelectatic rales at the apex, along the sternum and along the lower lung margins, will usually be removed with a few deep breaths. Muscle and fibrous tissue sounds may confuse, especially at the apex, as may also the crackles over the sternoclavicular and 1st rib joints. Passive movement of the shoulder while the breath is held will produce such sounds. Joint and cartilagenous sounds may be conducted to some distance from the sternum. Skin and hair crackles can be eliminated by greasing the skin.

In suggestive cases a period of observation is necessary, during which a record of temperature, pulse and weight will be made, the sputum collected for laboratory examination, and physical examination repeated.

Temperature should not be taken within half an hour of having had anything to eat, drink or smoke, nor of being out-of-doors. The thermometer should not be removed from the mouth until a period of from seven to ten minutes has elapsed. Small temperature rises, with which we are concerned, are often missed because too short a time is allowed. This obtains with any type or make of thermometer. Women will frequently show a premenstrual rise, beginning as long as ten days before the onset of menstruation.

The pulse should be recorded (after half an hour's rest) at the same time as the temperature. It is a more sensitive index of toxemia, but external influences are less easily avoided.

Weight is to be considered in relation to a standard for height, age and sex, and its fluctuations at noteworthy periods of the history.

Sputum from the chest should be collected. This may be coughed up at some one special period of the day, not necessarily the morning. Likely particles should be selected and a fairly prolonged search of slides may be necessary. The search for elastic tissue should not be forgotten in negative cases. One should

not be content with less than three negative examinations. The writer has found bacilli after twenty-one negatives, and knows of one case after sixty-three negatives. I have found bacilli of importance in making the diagnosis in 15 per cent. of cases; in the remainder it was simply confirmatory. Earlier examination might have made the finding diagnostic in a number of cases. There is need of much more bacillus hunting in the many cases of chronic respiratory disease in patients above middle age, especially for prophylactic reasons.

The X-ray should be regarded as supplementary and complementary to physical examination. It gives a great sense of security. No chest examination can be considered complete without an adequate reading of a pair of good stereoscopic plates, and a screen examination. Its revelations are often humiliating to even a careful examiner. Central lesions are revealed which are beyond the reach of the stethoscope and percussion. Compensatory emphysema may obscure more superficial pathological changes. Probably not more than one-fifth of all cavities are distinguished by physical signs. The X-ray will show a greater lesion in the majority of cases than can be demonstrated by physical signs. It is an historical record of many previous inflammations. The X-ray may be at fault with some minimal lesions, but it will help to a truer perspective in most cases, and it may point to some other cause than tuberculosis for the lesion. Especial caution is necessary in regard to the interpretation of shadows in children, possibly due to acute respiratory infections and to lymphatic absorption from the tonsils.

The screen is valueless and misleading in regard to an early diagnosis, but is useful and confirmatory for the grosser lesions.

Criteria for defining a diagnosis of tuberculosis are desirable, when B. tuberculosis has never been found, and for differentiating between active and inactive disease.

Symptomatology of Brain Tumors.

By C. A. WICKS, 3T0.

Cerebral tumors are unique in the nature and the variety of the symptoms they produce, in the diagnostic problems they present, and in the tragic disabilities with which they are often associated. While it is true that the accurate localization and the fine differential diagnosis lie in the field of the neurologist or the neurosurgeon, it is important that the general practitioner, the ophthalmologist and the aural specialist should be prepared to recognize certain symptoms which suggest the presence of a brain tumor. In this discussion, a brief review of the production of symptoms will be followed by a summary of several of the more common symptoms and signs exhibited by patients suffering from cerebral tumor.

Tumors produce their effects and thereby reveal their presence in the following manner:

(a) By disturbance of the normal functions of the brain and cranial nerves.

(b) By the production of an increased intra-cranial pressure.

The former mechanism takes place to some extent by means of pressure exerted directly upon the nervous tissue by the enlarging tumor, but chiefly by interference with the arterio-venous circulation to the part. This factor will be dealt with in more detail later. The latter condition (increased intra-cranial pressure) may be brought about by any one or more of three methods.

1. From pressure upon the walls of

the ventricles or inter-ventricular passages. In this manner, obstruction of varying degrees is offered to the flow of cerebro-spinal fluid from its place of origin in the choroid plexuses of the third and lateral ventricles to the subarachnoid spaces lying about the cortex of the brain. The presence of this increased amount of fluid in the ventricles constitutes the condition of internal hydrocephalus, and its effect will be to produce an increased intra-cranial pressure.

2. From the addition to the intracranial contents of a mass of constantly increasing bulk. Since, in any adult, the cavity of the skull has a fixed volume, then it follows that the addition of any extra body will raise the tension of the pre-existing contents.

3. From the chronic and occasionally acute oedema which exists about the advancing tumor and to a greater or less extent throughout more remote regions of the brain.

From a consideration of the anatomy and the physiology of the cerebro-spinal circulation, it will be seen that obstruction to the flow of cerebro-spinal fluid from the interior of the brain, can be most readily produced in the aqueduct of Sylvius and in the fourth ventricle. Therefore, a small tumor in the region of the inter-peduncular space, the corpora quadrigemina, or, in fact, at any point below the tentorium cerebelli, will comparatively early give rise to internal hydrocephalus with increased intracranial pressure. But now consider a tumor of the same size situated subcortically in either of the frontal lobes. Such a growth could have little or no effect on the flow of cerebro-spinal fluid. Not until it became much larger would it cause markedly increased intra-cranial pressure as the result of its bulk or accompanying oedema. The same reasoning might be applied to tumors of other regions above the tentorium cerebelli. Thus, supra- and intra tentorial tumors may usually be clearly differentiated. In the latter, symptoms of increased pressure develop rapidly, and when once established are persistent, whereas in the former, such symptoms tend to appear at a much later stage and are often mild and intermittent in character.

Let us now consider the effect of cerebral tumors on the local arterio-venous circulation. One of the first effects of the pressure exerted by such a gradually enlarging mass will be interference with the venous return from the region of the brain immediately adjacent. Congestion of that area will therefore be produced. As the tumor grows, and the pressure on the neighbouring tissues increases, the capillaries will be compressed. This will result in anaemia of the cells in the advancing margin of the growth. Consequently the picture is one of a spreading area of anaemia with a peripheral zone of congestion. Now, it is a clearly established law of cerebral pathology that congestion causes irritation and anaemia, paralysis. Therefore, if the process involves the cells of the cerebral cortex, the basal ganglia, or the cranial nerve nuclei, we should expect to obtain symptoms, first of irritation and later of paralysis of these cells. It is obvious that the nature of these symptoms will depend upon the function (neurological connections) of the particular region affected. Thus a tumor encroaching upon the pre-central gyrus of the left side would produce spasmodic jerking followed by increasing paralysis of certain muscles of the right side of the body. What particular group of muscles are affected would depend upon what functional area of this gyrus was involved (e.g., pressure on the gyrus at the vertex causing symptoms referable to the leg muscles). To cite another example, tumors in the region of the hippocampal uncus may be accompanied by hallucinations of smell which later give way to a gradually increasing anosmia.

The symptoms and signs of brain tumors, for purposes of description, are divided into two main groups.

(a) General. Those due to increased intra-cranial pressure.

(b) Local. Those due to disturbance of neighbouring cerebral tissue.

General Symptoms.

This group includes the classical triad of headache, vomiting and optic neuritis.

HEADACHE.—In a series of one hundred consecutive cases of verified brain tumors, analyzed by Dowman and Smith, this symptom was present at some time during the course of the disease in eighty one cases. In thirty-seven cases it was the first symptom noticed. The character of the headache is variously described. The pain is usually of a dull, diffuse type, but paroxysms of severe stabbing pain may occur. These paroxysms are often initiated by muscular effort, coughing, or other conditions which tend to produce active or passive cerebral congestion. With tumors above the tentorium, there are frequently long intervals of freedom from pain; but with infra-tentorial tumors the headache is usually constant and severe. In many cases, the pain is localized to a particular portion of the head, and with meningiomata over the vertex of the brain, localized tenderness of the scalp may be present.

VOMITING.—In the group of one hundred cases noted above, a history of vomiting was obtained in fifty-four. It is not usually preceded by nausea and therefore comes on as a sudden projectile act without warning. In a few cases, however, nausea occurred at some stage of the disease unaccompanied by vomiting.

OPTIC NEURITIS.—This condition was observed in seventy of the series of one hundred cases analyzed. Various terms such as papillitis, papilloedema and choked disk are used synonomously in the literature to designate the findings in the fundus of the eye in cases of increased intra-cranial pressure. The presence of optic neuritis can be definitely established by the use of the ophthalmoscope. In the early stages, the disk is pink, the edges blurred and the retinal veins engorged. Later, the disk may be quite red, its outlines obscured, the congestion more marked, and occasionally there are haemorrhages in the retina. The physiological cupping of the disk disappears co-incidentally with its bulging forward from the general surface of the retina. This latter fact necessitates the insertion of more convex lenses in focussing from the retina to the top of the optic disk. The difference in these focal lengths, expressed in diopters, is given as a measure of the degree of papilloedema. Thus a choking of less than three diopters (1 mm.) is considered a moderate finding, while marked choking may give readings as high as ten diopters. Vision may be unimpaired, even with severe optic neuritis; and moderate papilloedema often exists for years without any gross disturbance of either visual acuity or field of vision. During this time the patient is entirely unaware of any abnormality in the sense of sight. For this reason, ophthalmoscopic examination should not be restricted to those cases in which the patient complains of symptoms definitely referable to the eyes. Furthermore, this relatively simple clinical procedure is certainly indicated in all cases presenting obscure symptoms suggesting cerebral disease.

If the increased intra-cranial pressure is not relieved, so that the optic neuritis persists, secondary optic atrophy eventually develops. This condition is accompanied by blurred vision and contracted visual fields. The fundal picture now shows the disk to be grey, with irregular outline, and the retina pale with contracted arteries.

Other symptoms of increased intra-cranial pressure are: Dizziness, Drowsiness, generalized Convulsions, and transient attacks of Diplopia.

In fifty-seven of the one hundred cases, a condition indicative of increased intra-cranial pressure, was the first abnormality observed. Headache was the initial complaint in thirty-seven

cases, convulsions in ten, blurred vision in four, dizziness in three, vomiting in two and mental dullness in one. One must not, of course, draw too sweeping conclusions from the above figures, owing to the relatively small number of tumors considered.

Localizing Symptoms.

It has been the custom to consider the local symptoms of brain tumors from the point of view of their anatomical position (e.g., in the various lobes, etc.) The subject is extensively treated from this angle of approach in neurological text books and monographs. The procedure here, will be, first to mention a common localizing symptom and then to give some rough indication of the probable situation of the tumor.

JACKSONIAN EPILEPSY. — This symptom is to be distinguished from the generalized convulsions which may occur, with an increased intra-cranial pressure: but also in many cases where this pressure is normal the mechanism here being rather obscure.

In typical Jacksonian epilepsy, tonic contraction quickly followed by clonic spasms begin in a particular group of muscles and extend rapidly to contiguous gronps of muscles. There is usually no loss of consciousness in these attacks. Preceding the initial twitching of the muscles there is often a so-called "signal symptom." This may take the form of a tingling or numb sensation in the area to become the seat of the spasm. Following the seizures, there is usually a definite weakness of the muscles affected. The above symptoms indicate irritation of the cells of the motor cortex, and the particular muscles taking part in these spasms will be determined by the area of the motor cortex involved. Thus, a tumor pressing upon the upper end of the pre-central gyrus would produce twitching of the leg muscles on the opposite side of the body. Similarly, with pressure on the lower portion, near the Sylvian fissure, epileptiform attacks in the muscles of the contra-lateral side of the face would result.

PROGRESSIVE MUSCULAR Weakness.—This may be associated with the occurrence of Jacksonian epilepsy, as noted above, or it may be the only symptom present. In the former, the growth is involving the cortex of the motor area of the brain, and the paralysis, in this case, is usnally of the monoplegic type. In the latter case, however, the tumour is probably pressing upon the motor fibres in the internal capsule, or as they are converging upon that structure. With this condition, the paralysis rapidly becomes hemiplegic. Tumours of the cerebral peduncles, pons and medulla may also give rise to hemiplegia, complicated by marked general symptoms as well as signs of cranial nerve involvement.

VISUAL DISTURBANCES.—A loss of visual acuity eventually occurs in cases of long standing optic neuritis, in the manner already described. Tumors in the region of the lower visual centres, the visual cortex or at any point along the path of the optic impulses, will also produce defects in the patient's sight. When bright flashes of light, often coloured and suggesting fireworks are followed by periods of blindness, a cortical tumour of the visual area is indicated, and signifies irritation of the cortical cells of that region.

A careful examination of the visual fields by means of the perimeter is essential in the localization of growths along the lengthy course of the fibres carrying the visual impulses. For example, a chart showing bi-temporal hemianopia suggests the presence of a pituitary tumour pressing upon the mid-portion of the optic chiasma. To cite another instance, a lesion in the lower part of the right temporal lobe will involve those fibres which are conducting impulses from the lower segment of the right half of each retina, and therefore will produce loss of sight in the left upper quadrants of both visual fields; (i.e.—left upper quadrantic homonymous field defect).

SENSORY DISTURBANCES.—Disturbances of sensation in cases of brain

tumor are usually in the nature of loss of the sense of posture, of localization of touch and of the perception of degrees of heat and cold. Astereognosis, or the inability to recognize the shape of objects by touch, is sometimes a striking feature. The above symptoms may be associated with a tumor of the parietal lobe which is pressing forward into the post-central gyrus. Growths in the medulla, pons, peduncles and internal capsule might also account for such loss of sensation by pressure upon the sensory fibres descending from the post-central gyrus. Marked general symptoms are usually present in these latter cases.

MENTAL CHANGES.—We have already seen that stupor and drowsiness can be produced by increased intracranial pressure. General dulling of the intellect, change in character, and dimming of the memory may result from lesions of the so-called "silent area" in front of the pre-central gyrus. Often, however, the first indication of such a tumor will be signs of involvement of the motor cortex. This is especially true of the right frontal lobe which appears to be less important functionally than the corresponding lobe on the left side.

MOTOR APHASIA.—In this condition, the individual knows the words he wishes to use and can look them up in a dictionary, but is incapable of producing the proper sounds. This symptom would indicate a lesion in the region of Broca's area which, in those who are right-handed, is situated in the left inferior frontal convolution.

VERTIGO.—As a localizing symptom of brain tumour, vertigo suggests disturbance of the structures about the cerebello-pontine angle. Growths in this position are often associated with staggering gait, nystagmus and unilateral nerve-deafness. This symptom complex is typical of acoustic neurinomas.

The above list of signs and symptoms is not intended to be complete, but a sufficient number have been mentioned to illustrate the type of condition which should lead us to suspect the presence of an intra-cranial tumour and should entitle the patient to a further investigation with that possibility in view.

Blood Chemistry in Relation to Clinical Medicine.

By GEORGE HUNTER, M.A., B.Sc. (Glasg.),
Assistant Professor, Department of Pathological Chemistry.

The Emergence of a New Adjunct to Medicine.

If we recall that blood chemistry in its present relationship to clinical medicine has a very short history, certain difficulties in the relationship of the physician to the so-called "laboratory man" become explainable. For it would appear to the writer, who is in the latter category, that many laboratory men without medical degrees in American hospitals have for the past half dozen years or so, been virtually diagnosing the disease and prescribing the treatment, as well as providing the blood analysis. But if we agree that blood chemistry entered medicine with the introduction of the Folin and Wu system of blood analysis only in 1920-22,* then the situation is not surprising. The methods almost without exception have been elaborated by men without medical

* I am aware that in 1821 Prevost and Dumas found an increase in the non-coagulable nitrogen after nephrectomy, that Bright in 1834 showed that it was raised in kidney disease, that Strauss in 1902 showed that the **Reststickstoff** in normal serum varied between 20 and 55 mg. and that almost half of this was urea nitrogen; that Garrod made approximate estimations of the uric acid of the blood in gout about eighty years ago, that blood sugar and acid base balance likewise have histories stretching back long before this time but nevertheless it is true as a general statement that the introduction of tungstic acid as a protein precipitant, also marks the date of the introduction of blood chemistry as a routine clinical procedure.

qualification, and the main ones now in use published within a few years of each other, with the result that the medical world was taken unawares. Having great faith in the scientific method, and this particular one did not want for too enthusiastic advocates, the doctors as a class rather fell a prey to the laboratory man. But how could they avoid it? The field was completely new to medicine, the doctor could not judge when a blood analysis might be of assistance in diagnosis or treatment, and if he did obtain from the laboratory man a "routine blood analysis" (something quite meaningless, but the best he could ask for) then he was not in a position to interpret whatever results he had handed to him—without the assistance of the laboratory man. But we cannot conclude that the laboratory man was the villain of the piece or the medical man his dupe; the rather ridiculous situation was merely a manifestation of the emergence of a new adjunct to the medical profession, a likely enough phenomenon in these evolutionary days. For a moment the tail may have been mistaken for the dog, but the true relationship of blood chemistry to medicine is now gradually becoming clear to the doctor.

My attitude thus defined, I shall not presume to trespass on the territory of the medical teacher or practitioner who is now conscious that his marches need attention, but shall confine myself to the laboratory aspect. The time is now past for any reiteration of the importance of blood chemistry in medicine. It is for the physician to decide its importance in specific pathological cases, but it is now unquestionably an established and necessary adjunct to medicine. Medical literature of the past six years, especially on diabetes and nephritis, must have been hard reading for the practitioner who never heard of blood urea, non-protein nitrogen, creatinine, or CO_2 combining power; and the medical student who has the advantage of making such determinations for himself in the course of his training is merely fitting himself for the new medicine.

Will the Doctor do his own Blood Analysis? The question may be raised at this point, for it is in the student's mind in this connection, of what blood analyses he is likely to be able to do in his own office when he becomes a practitioner. In the first place it may be noted that blood analyses are mainly of value in severe pathological conditions, and can be but relatively seldom needed in general practice. Some of the reagents used are unstable, and apparatus quickly gets dusty and needs relatively much more attention when not continually used. Then there is the personal factor. If a man has a bent towards, or an interest in this type of work he may get good results, but if he has not, then his results are not likely to be reliable. A good colorimeter is an expensive item and colorimetric determinations without one are not dependable. At most it would appear that a doctor might determine his own blood sugars by the Shaffer-Hartman method and urea nitrogen by urease and aspiration, as the apparatus and reagents for these are both inexpensive and simple. But it should be further noted that the tendency towards extreme simplification of method for clinical purposes is one which has even now perhaps overshot the mark. The methods of the Folin and Wu system are practically as simple as it is possible to make methods compatible with any degree of accuracy, and the amounts of blood required for them are not regarded as too great from the practitioner's point of view. So there is little call to adapt the methods to the quantity of blood obtainable, for example, by finger prick. It would appear from these considerations that there is little likelihood that the physician of the future will concern himself to any great extent with blood analysis. The apparent difficulties of the situation are already being solved in the cities by groups of physicians having laboratories where a trained technician may do such work for

the group, and in the country by the establishment of similar services in the local hospitals.

Blood chemistry is now, however, an integral part of the medical student's training as are bacteriology, opthalmology or radiology. The subject is, as already indicated, still in the "emergent" stage, and for this reason it is the purpose of this article to give the reader a bird's-eye view of the new field.

Taking Bearings.—A student who has "taken" blood chemistry may perchance in his search after truth be studying the composition of blood in such a text-book as that of Mathews on physiological chemistry, and has out of mere curiosity totalled the percentages of constituents in the analyses there given by Carl Schmidt and by Abderhalden. He will have found, perhaps to his astonishment, that Carl Schmidt's analysis tallies with the required total even to the second decimal place, and that, without mention of glucose, urea, non-protein nitrogen, uric acid or creatinine. His analysis was, however, made in 1850, and for this reason the student may regard it as out of date and think Mathews should not have included it in a modern text-book. If a few pages further on be total Abderhalden's constituents in 1,000 gm. of ox blood he will find that 999.792 gm. are accounted for, glucose being the only constituent above mentioned, included. That is, the percentage discrepancy is 0.0218; or 21.8 mg. in 100 cc. is left as the maximum material for the study of what we presume to call "blood chemistry."

As a matter of fact the analysis are not strictly accurate, but they serve the purpose of emphasizing that the non-mineral non-protein, or more positively the organic non-protein constituents of blood, in which clinical medicine is now specially interested, constitute in reality a relatively small proportion of the total blood. We thus speak of glucose, for example, as being present in blood in relatively large amount at a concentration of 0.1 per cent. Normal amounts of constituents which have proved of significance in clinical medicine, are urea, 25 mg., uric acid, 3 mg., creatinine, 1 mg., and bilirubin, 0.1 mg. per 100 cc. The total of these is only about 30 mg. There are, however, many other substances whose behaviour has not been studied with relation to medicine, which come into this non-protein organic fraction, among which may be mentioned glutathione, present in human blood to the extent of about 50 mg. per 100 cc., substances found in large amounts in muscle, such as creatine and carnosine, the former amounting to about 4 mg. and the latter to perhaps half that concentration; in addition to which are present about a score of the free amino acids. The latter are present in small amounts and an idea of their concentration in plasma is obtainable only from the fact that its total amino nitrogen is about 5 mg. per 100 cc. Taking this as 10 per cent. of the actual amount of amino acids, there would then be approximately 50 mg. of amino acids per 100 cc. of plasma, assuming their presence in like concentration. That is a maximum estimate, as there is reason to believe that part of the amino nitrogen obtained by present methods of determination arises from peptides. I could thus proceed to take the individual known urinary nitrogenous constituents and the constituents of muscle and discuss what we know of their presence in blood, but it would not further the purpose of this article. Let us pause for a moment and consider the total amount of these main nitrogenous constituents. They amount in all to about 136 mg. per 100 cc. of blood. Add to this the non-protein non-nitrogenous organic constituents, glucose 100 mg. and cholsterol 150 mg. and we still have something less than 300 mg. or 0.3 per cent. of the blood constituents accounted for. There are many other substances of this kind known to be present in blood and many others unknown, but their amounts are relatively so small that the sum total

of organic non-protein substances of blood cannot well exceed 0.5 per cent.

Expressed in these terms the reader may get some idea of the field in which clinical blood chemistry is chiefly occupied, but may expect some apology for its apparent narrowness. It may be pointed out, however, that blood in this respect is like a precious metal ore. An ore running $10.00 a ton contains perhaps less than 1 mg. of gold per 100 gm. of mineral. Or, expressed in a different way, this very small fraction of the blood certainly holds over 90 per cent. of the total number of constituents.

Delicacy of Methods.—Arising out of the relatively small amounts of blood available from patients, and from the low concentrations in that blood of the constituents we want to determine, the methods of analysis must be very much finer than those in use in general chemical analysis. Gravimetric methods are quite out of the question for routine blood analysis. Titration methods outside of iodimetry, which is applicable in relatively few instances, are also insufficiently delicate, so that the future lies almost exclusively with colorimetric measurement. In a few instances the spectroscopic method may be useful and in some also the physiological assay is the only available one, but neither of these need here be considered, as they are not strictly chemical. The titration method for urea, with the use of urease, is one of the least delicate methods employed in clincial chemistry and the relatively large amount of 3 cc. of blood is required. In a blood containing 30 mg. of urea per 100 cc., we thus use in the analysis 0.9 mg. of urea. In the Shaffer-Hartman method for blood sugar the equivalent of 0.2 cc. of blood is used and this contains normally about 0.2 mg. of glucose. Uric acid is estimated satisfactorily on the equivalent of 0.5 cc. of blood in which the amount of uric acid present is about 0.015 mg., but we must take bilirubin to get a more accurate idea of the extreme delicacy attainable by the colorimetric method. We have in normal human serum about 0.1 mg. of bilirubin per 100 cc., yet this constituent can be estimated by coupling with diazotized sulphanilic acid with a fair degree of accuracy in 0.5 cc. of serum or plasma. The amount of bilirubin used in such an estimation is of the order of 0.0005 mg. or one two-millionth part of a gram.

Expression of Results.—A word may be said about the expression of the results of blood analysis. The most useful method for such constituents is undoubtedly in milligrams per 100 cc. of blood. On general principles uniformity is not a particularly inspiring ideal, but in instances of this kind departure from it is mere contrariness. 100 cc. is the basis accepted by all the best workers on blood chemistry and there seems to the writer no reason whatsoever why sugar should be expressed by certain clinical workers on the basis of 1 cc. or bilirubin on the basis of 1000 cc. of blood. Such a procedure merely leads to confusion, and distorts in the mind of the reader the chemical blood picture. The case of bilirubin is particularly instructive from this point of view. Van den Bergh, who first introduced the diazo method for its quantitative determination employed (translated) the term "unit," which is still the most common in the literature of this subject. His unit was defined as one part of bilirubin in 200,000 parts of serum, which works out at 0.5 mg. per 100 cc. This leads to further confusion, as a unit is two to five times greater than the concentration of bilirubin in normal human serum. It would seem to the writer that the term "unit" should be confined for want of a better means of expression, to, for example, physiological assay work, and that there is no justification for its continuance as a measure of such a substance as bilirubin, which is obtainable in the pure state. And it would lead to less confusion if we regarded the normal for human serum as 0.1 to 0.3 mg. per 100 cc. of blood. Such an expression of result is, of

course, obtained by simply halving the Van den Bergh unitage.

Applications.—The three main pathological conditions in which the organic non-protein constituents are of clinical significance are nephritis, diabetes mellitus and jaundice.

In nephritis the physician has a choice of any or all of:—urea nitrogen, non-protein nitrogen, uric acid and creatinine. The estimation of uric acid in this condition has indeed been largely abandoned for the reasons that it gives no more information than the others, other factors besides the kidney affect its level in the blood, and thirdly, the commonest clinical method for its estimation is not reliable. Most people apparently agree that urea nitrogen and non-protein nitrogen give approximately the same information in advanced chronic nephritis, but it should be borne in mind, despite the preference of many for non-protein nitrogen in contradistinction to urea nitrogen, that some of the non-protein nitrogen arises from constituents such as amino acids, glutathione, etc., the level of which in the blood is not affected even in advanced retention of the common waste products. For this reason the urea nitrogen is a more specific index of the degree of retention than is non-protein nitrogen.

In diabetes the typical findings are the result of a deficient production of insulin by the islets of Langerhans in the pancreas. This results in a disturbance in carbohydrate, and secondarily, in fat metabolism reflected by hyperglycaemia and frequently by the presence of ketone bodies in the blood, but without significant changes in the nitrogenous substances of interest in nephritis. A satisfactory method for the determination of blood sugar was one of the greatest factors in the discovery and study of insulin and the determination of blood sugar must remain an important procedure in the study and treatment of the diabetic with insulin.

Next in importance to nephritis and diabetes comes perhaps jaundice. The diazo test for bilirubin has proved of great value in the estimation of bilirubin in the blood serum in a variety of clinical conditions, but above all has been a great stimulus to theory on different types of jaundice and to the physiological study of bilirubin. According to the diazo test there are two types of bilirubin, one giving a so-called "direct," the other only an "indirect" reaction, found respectively in sera in obstructive and in haemolytic jaundice. Whatever clinical opinion may now be, with regard to the practical use of the diazo test to distinguish those conditions, there is no doubt that there are two types of bilirubin. The obstructive type is dialysable, the hæmolytic is not, corresponding to the general finding that there is bilirubinuria in the former condition, but not in the latter. They differ in solubility and precipitability, but there has not been any satisfactory explanation to account for the two types. Haematoidin, which has long been of interest to histologists, has now been proved to be identical with bilirubin, but it would appear to me probable that there is just sufficient difference between them to account for the different types of diazo reaction.

Other Clinical Methods. — The CO_2 capacity of the serum, which is a clinical method outside the field of the non-protein organic constituents we have been discussing, is the simplest means of determining the danger imminent to the patient from acidosis or alkalosis. The biochemical literature arising from the study of the physico-chemical aspect of blood is now very great and the mass of theory imposing in its exactitude and completeness. It is practically complete in many respects without consideration of the non-protein organic constituents, not because the latter are of no importance, but because their concentrations are too small to affect materially the physico-chemical model shaped by the major blood constituents such as protein and inorganic salts. Much mathematical treatment is necessary in the exposi-

tion of acid base equilibrium, hydrogen-ion concentration and related aspects, but the practical implications of a low or high CO_2 capacity are simple enough and clearly enough explained in modern text-books of biochemistry.

I can refer to the determination of other blood constituents merely as a miscellaneous group. There are now few of the known blood constituents for which at least approximate methods of determination do not exist. Some of these necessarily require much technical skill and are unsuited as yet for routine work. Apart from methods for the determination of haemoglobin and the other blood proteins, we may mention those for fat, lecithin and cholesterol and for the inorganic constituents such as calcium, magnesium, sodium, potassium and phosphorus, chlorides and sulphates.

Finally I may remark that blood still remains a fertile field for investigation. Just what lines of attack are likely to yield the richest harvest is a matter of personal predilection, but the largest unknown field is yet in all probability that which has had the greatest attention in this article, the small field of organic non-protein substances. Its existence in the systematic analysis of blood is lost sight of, and the modern physio-chemical picture largely dispenses with it, so that between these two influences it has perhaps not had the attention it deserves. The rationale of blood chemistry, from a metabolic aspect, rests on the assumption that chemical changes in the organs and tissues will be reflected in a changed composition of the blood. The assumption has been justified in many instances, and if we can also point to many where it has not, these failures are explainable by the inadequacy of present methods to detect or determine the minute amounts of substances which we might be led to look for in particular pathological conditions.

Duodenal Ulcer.

By V. I. E. HENRY, 3T0.

Duodenal ulcer is so closely associated symptomatically with gastric ulcer that they are usually described together under the heading of peptic ulcer, but about 60% of the so-called peptic ulcers occur in the duodenum. 95% of these ulcers are found within one inch of the pylorus, usually on the upper or anterior wall of the duodenum.

Duodenal ulcers occur in both chronic and acute form—usually they are single, but in 8.2% of the Mayo series multiple ulcers were found. The acute ulcer is more common in females than in males, and is apt to occur at any age —often being a complication of an acute fever. About 6.2% of skin burns coming to autopsy show acute duodenal ulcers. The chronic ulcer is more common in males than in females—the age incidence is about 15-25 years in females, and in males, 30-50 years. Some authorities believe that a relationship certainly exists between chronic appendicitis and peptic ulcers.

The acute ulcer is usually small, punched out, with clear-cut edges and a smooth base, and the peritoneal surface is not thickened, whereas the chronic ulcer is larger, and has an irregular border and indurated edges.

The etiology of peptic ulcers, as they occur clinically, has not, as yet, been satisfactorily established in all particulars, but two essential factors are recognized:

(1) Localized malnutrition or necrosis involving the mucous membranes of the walls of the stomach or that part of the duodenum subjected to the di-

gestive action of the gastric juice. Bolton showed that it was merely necessary for the cells to be damaged to some extent.

(2) The digestive action of the gastric juice.

The vitality of a limited area of mucosa may be impaired by interference with the local blood supply, e.g., by embolism or thrombosis. The coincident occurrence of acute alveolar abscess, tonsillitis, etc., with the onset of ulcer symptoms, has led to the belief that focal infection may play an important part in the development of peptic ulcer. Embolic bacterial invasion, as a factor in producing ulcer has been demonstrated by experiments on animals, and Rosenow showed that "streptococci irrespective of their source when of a certain grade of virulence exhibit affiinity for the gastric mucous membrane—when injected intravenously, ulcer of the stomach and duodenum may follow." That anaemia, worry and nervous strain are potent factors in disturbing the digestive function, exciting pylorospasm and playing a detrimental role in trophic control and circulation, was shown by Bevan—who found that the incidence of peptic ulcer in Central Europe was markedly increased during the last war.

The symptoms associated with peptic ulcer are quite variable both in type and degree. Ulcers have been found post mortem in cases where no symptoms have been noted during life. The following symptoms are most constantly present:

1. Epigastric pain which
   (a) appears at a considerable time after meals.
   (b) is associated with increased free hydrochloric acid in the stomach.
   (c) is relieved by taking food or alkali.
   (d) is usually increased by activity and decreased by rest.
   (e) may be partially or completely relieved by vomiting.
2. Nausea and vomiting.

They are not constantly present, but are more frequently present when the epigastric pain is severe or when there is pyloric obstruction complicating the ulcer. The vomitus may or may not contain blood — only in about 1% of ulcers is there recognizable hematemesis.

3. Hemorrhage occurs in about 33% of cases. This bleeding may be latent or profuse.

4. Tenderness on pressure with increased resistance is common, the situation being more or less constant in the same individual.

5. Hypersecretion of the stomach—which is symptomically indicated by "water brash."

The diagnosis can usually be made on the history, and is confirmed by X-ray examination. X-ray, as a rule, shows a defect in the contour of the duodenal caput, and in cases complicated by obstruction there is a delay in the emptying of the stomach.

The following cases are of some value in illustrating the type of history obtained—Case I. R.T. 24-5-28.

"A male patient of 52 years, who denies having indigestion except for an occasional gas attack lasting a week or so—until 1925. In the spring of 1925 about 5 a.m. he was awakened with excruciating abdominal pain and called a physician, who sent him forthwith to hospital, where he was operated on and a duodenal ulcer sutured. He remained in hospital 2½ months. He followed no dietary course after leaving hospital—no digestive disturbance occurred for two years. Last spring he began to have a dull, gnawing pain about the umbilical area, coming on 1 or 2 hours after meals. This pain has been present every day since. Food and soda give relief—occasionally there is night pain. He has no qualitative food intolerance, appetite is good—no vomiting, no melaena—no jaundice—no "clay" stools. There is a weight loss of about 20 lbs.—somewhat constipated."

This patient was placed on peptic management, and after 3 months left the hospital "80% better," but still had pain and belching of gas after meals, but it was not very severe. He was unable to follow a diet outside of hospital, hence returned after 3 months for further treatment.

6-10-29. "His symptoms are the same, slight pain 2 to 3 hours after meals, with belching of gas. He never is nauseated nor vomits. No passage of blood in the stool or hematemesis.

X-ray examination summary. The examination is negative for gastric ulcer and cancer, and positive for duodenal ulcer.

Note: This patient has in addition to the chronic duodenal ulcer, cirrhosis of the liver and erythromelalgia.

Case II. E.R. 16-1 29.

A male patient of 45 years, who complains of a heavy feeling in the epigastrium after meals, occurring perodically during the last 5-6 years. This distress occurred about 15 minutes after meals, lasting 2 to 3 hours, and was relieved by "dyspepsin pills."

There were no other symptoms until Jan. 13th, when on his way to work, patient felt faint, perspired freely, but did not lose consciousness. After a light breakfast he went home, but did not go to bed. After lunch of bread and milk he had another "dizzy spell," but was relieved by taking spirits of ammonia. The next day he got up, had a complete breakfast, but did not go to work. Later be had a light lunch, which was followed by belching gas, and in about 30 minutes by a "dizzy spell" with hiccoughs. Three hours later he vomited a ½ pint of brownish material and fainted. He slept well that night, but felt weak the next day, so remained in bed. He had no epigastric pain, but there was belching of gas after meals. He was admitted to hospital that night and on examination occult blood was found in the faeces and blood examination showed:

R.B.C. 1,200,000.
W.B.C. 8,200.
Hb. 46%.

X-ray examination summary: There are defects noted in the duodenal caput, the appearance of which would indicate the presence of ulcer."

This patient has probably had a chronic duodenal ulcer for 5 or 6 years, from which haemorrhage occurred Jan. 13th.

Unless there are complications such that necessitate immediate surgical interference, e.g., perforation, it is wise to try medical treatment first in cases of peptic ulcer. Should these ulcers not react to medical treatment or should they be complicated by recurrent hemorrhages, perigastric adhesions or obstruction, it is then necessary to apply surgical measures. Theprinciples of medical treatment are:

1. The control of foci of infection, especially those occurring in the mouth.
2. The obtaining of gastric rest—as complete as possible.
3. Neutralization of acidity.

This is most often carried out by the use of the Sippy method or some modification of it. The patient is put to bed for several weeks, fluids restricted, and food given q. 2 h. x 8 in the form of a mixture of milk and cream. The quantity per feeding is at first 3 oz. and is gradually increased to 6 oz.; alkali is given between each feeding in the form of a powder containing gr. x. each of heavy calcinated magnesia and sodium bicarbonate, alternating with a powder of gr. x. of bismuth carbonate and gr. xxx of sodium bicarbonate. After about two weeks the milk may be supplemented with soups, custards, soft boiled eggs, etc., and gradually the diet is increased to the required caloric intake The milk feedings are first replaced by five small meals, which are eventually cut down to three moderate meals. Some advocate the taking of a glass of milk between meals, and at bedtime, to utilize the secretion of the

stomach; and by this means, dispense with the administration of alkali, as soon as possible after the period of active treatment.

The bowels are regulated by the use of mineral oil, cascara or milk of magnesia. Drugs should be discontinued as early as possible.

The pain of ulcer is usually relieved by rest, diet and alkalies, but atropine may be given. If atropine is inadequate, codein (gr. ¼-½) is given and in very severe cases morphia (gr. ¼).

If the ulcer is complicated by haemorrhage the essential point is to put the patient at rest as quickly as possible. This is best obtained with opium. Such haemorrhages are rarely fatal, but may be sufficiently severe to cause fainting. Fluids may be administered interstitially or per rectum, and in severe conditions, serum intramuscularly or blood by transfusion. In these cases it is useless to try to control the haemorrhage by the administration of drugs by mouth.

Case II. was put to bed, fluids restricted and treated similarly to that outlined, with the result that on Jan. 27th he had no epigastric distress, no belching of gas after meals, no vomiting and no occult blood was found in the stools. Blood examination:

R.B.C. 3,200,000.
W.B.C. 12,500.

The after care following medical treatment is just as important as after surgical interference, in the case of ulcers. It is essential that the individual follow a simple regime, eat slowly and be careful to masticate his food thoroughly. The taking of alkali should be continued for several months, and thereafter taken at the first sign of discomfort. Some physicians advocate the taking of a glass of milk, instead of alkali, to relieve recurrent symptoms—in this way dispensing with drugs.

Testicular Swellings.

Gordon S. Foulds, M.B., M.S.

Scrotal swellings may arise from the testicle and epididymis, or from their coverings. For the purpose of this review, I shall only consider those of the former group. These swellings may be discussed under headings of Trauma, Inflammations and New Growth.

Trauma.

Owing to the free mobility of the testis and the protection of the thighs, severe injury of the testicle is not common. The most common injury is when a man falls astride an object. But in this case, damage to the urethra frequently overshadows that of the testicle. Severe contusions to the organ cause extreme pain of a sickening character, and are often associated with considerable shock. Haemorrhages may take place into the tunica vaginalis, the epididymis or the testicle proper, and in any case, there is usually a good deal of ecchymosis of the scrotum. The injury may be followed in many instances by a fairly acute epididymo-orchitis. Treatment consists of rest, support of the part and the application of ice packs.

Rotation of the testis upon it long axes, causing a twist in the cord, is known as torsion of the testicle. As a result of the twisting of the cord, the blood supply is interfered with to a varying degree. Torsion may occur with extreme exertion or may occur during sleep. It is accompanied by severe pain, moderate shock and often nausea and vomiting. The testicle becomes very tender and swollen. The condition is most often confused with either an acute epididymitis or a

strangulated hernia. The absence of an acute urethritis and the sudden onset in the first instance, point to torsion rather than epididymitis, while examination of the original ring together with the absence of any previous appearance of hernia, will rule out strangulated hernia.

The treatment of torsion consists of undoing the twist in early cases, while in late cases where considerable damage to the testis has taken place, orchidectomy may be necessary.

Inflammations.

Inflammatory conditions arising in the testis and epididymis may be divided into acute and chronic, and these two sub-divisions again be sub-divided, the acute into gonorrheal and non-gonorrheal . The chronic may be divided into simple chronic and tuberculous epididymitis, and syphilitic orchitis.

The acute inflammations involving the epididymis, in patients suffering from gonorrhea, is the result of retrograde infection down the vas deferens, and consequently, primarily involves the globus minor first. In more acute forms, probably this stage is not seen as the whole epididymis becomes acutely swollen. The swelling is further increased by a small secondary hydrocele, which may extend so that it is difficult to palpate the epididymis separately from the testicle. A diagnosis of this condition is easy, as the patient himself will usually volunteer information with regard to his acute urethritis. Treatment of this condition consists of supporting the part on a suitable bridge of adhesive plaster across the thighs, rest in bed and the application of ice packs. This constitutes the usual routine therapy. Where possible, diathermy to the inflamed part with Corbus clamp is very satisfactory. There is probably no condition in urology where medical diathermy is more applicable. It should be applied for thirty to forty-five minutes each day, the temperature being maintained as high as can be comfortably borne. The pain will frequently subside after the first or second treatment. This greatly shortens the time of incapacity from the condition.

Non-specific epididymitis usually occurs in older men, particularly in those subject to prostatic or other urethral obstructions, most frequently after instrumentation which has been rough or where proper aseptic precautions have not been taken. Physical findings are as a rule, similar to those in acute epididymitis of gonorrheal origin. The age of the patient with a history of inflammation and lack of any history of urethral discharge, will usually suffice to make the nature of the condition clear. Treatment may be commenced in a similar fashion to that already outlined for the gonorrheal inflammation, but the result will be different in the majority of cases. It is extremely rare to find gonorrheal epididymitis suppurate. Whereas, in the non-specific epididymitis, the infecting organism is most frequently colon bacillus or staphylococcus, and suppuration is the rule rather than resolution. The part must be watched and examined from day to day and when definite fluctuation follows signs of suppuration, an incision should be made over the softest point. A single incision may be sufficient in the majority of cases, but where a pocket of pus is large, it may be wise to obtain dependent drainage by a through and through incision from this point to the most dependent part of the pocket.

Simple chronic inflammatory conditions of the epididymis are not at all common, except as a residual finding after a preceding acute infection.

Tuberculosis of the epididymis is probably the most common of the primarily chronic inflammations occurring in this organ. The mode of infection is a keenly debated one. There are two accepted routes of infection. Some observers claim that the infection reaches the epididymis by way of the vas deferens and that the prostate or seminal vesical is the primary lesion so far as the genital system is concerned. This

view is held by Young and his followers. The view that the infection in the epididymis is hematogenous is more widely accepted amongst urologic surgeons. In recent writings this view is ably supported by Barney of Boston.

The history in tuberculous epididymitis is usually that of a tender or slightly painful nodule appearing in the upper pole of the epididymis. This gradually and slowly progresses until the whole epididymis is distinctly enlarged, thickened and nodular. On examination, in addition to these findings, the vas deferens is irregularly thickened, giving a beaded feeling. On rectal examination, the prostate is irregularly thickened and nodular, firm, but not hard, while the seminal vesical on the affected side, is often irregularly thickened. The accepted treatment of tuberculous epididymitis is epididymectomy. The frequency with which the disease recurs on the opposite side makes it important that a complete orchidectomy should not be done, unless absolutely unavoidable. As in the treatment of surgical tuberculous processes elsewhere, it must be remembered that the condition is frequently not a primary one, and that there are probably, if not invariably, other tuberculous lesions, possibly quiescent, present. Consequently, the patient must be kept on a careful general treatment for tuberculosis for some time.

Syphilis involves, not the epididymis, but the testicle itself. The testicle becomes enlarged, retaining its normal shape, is firm and smooth. The epididymis can be felt to be normal in size and consistency and uninvolved in the process. A characteristic finding in this condition is the lack of normal testicular sensation. The Wassermann reaction will be found to be positive and the condition will yield to an anti-syphilitic treatment.

New Growths.

New growths in the testicle arise with rare exception, from the sex cells, situated in the rete testis. The main characteristic of these cells is that they are toti-potent, and may therefore, develop tissue recognized as a derivative of any of the three germinal layers. Large polyhedral cells of embryal type may enter into the picture. The most frequent single type of teratoma is of this nature. While cells are potentially capable of producing any or all of the tissues derived from the three germinal layers, resulting tumors are usually found to consist of some one predominating element.

The condition must be differentiated from the several foregoing types of testicular swelling. The patients are usually men under forty years of age, who complain of swelling of the testis. There is seldom any associated pain. Other symptoms, such as loss of weight, and lumbar backache, are not uncommon. The testicular sensation is frequently diminished, but in these patients the Wassermann reaction is negative. The normal testicular outline is retained, but the tumor mass is stony hard and somewhat irregular in the surface. Treatment of tumors of the testis is a combination of conservative surgical interference combined with X-ray and radium therapy. On account of the fact that the lymph drainage from the testes follows the spermatic artery until it crosses the ureter and then enters the lumbar glands, it makes it impossible to safely remove the gland field. Consequently, orchidectomy should be done, care being taken to clamp the cord before manipulation of the testicle itself is undertaken. Following orchidectomy, careful and thorough X-ray treatment should be instituted, having particular reference to the lumbar lymph glands.

A Report on a Fatal Case of Coronary Thrombosis.

By F. F. FITCH, B.A.

The following is a brief resumé of a case sent into the Toronto General Hospital with a diagnosis of "Perforated Duodenal Ulcer," and emphasizes very well the importance of a complete and accurate history as well as the correct interpretation of physical findings.

A. J. S., age 69, a blacksmith, worked at his trade until five years ago, when he was in a street-car accident. He gave a typical history of duodenal ulcer for the last five years—a history varying from the classical picture only in the absence of the seasonal periodicity, in all other respects perfect. The first attack of pain left him nauseated and faint, but did not cause him to vomit. The pain, griping and colicy, would last one to two weeks, would be relieved by food or alkalies, and come on a short time before each meal and about 3 o'clock in the morning. He always carried biscuits with him and would obtain through them relief until the next meal. There would be remissions of the pain for varying lengths of time. This pain was always situated in the epigastric area and the last attack occurred on December 23rd and was relieved by food. Five days later he took some castor oil which gave him three or four free movements and at the same time caused his pain to entirely disappear, leaving him perfectly well for the next three days. On January 6th when he arose at 5 a.m. to turn on the light in the chicken house he experienced a slight aching pain high up in the epigastrium in the midline. He returned to bed as usual until his customary rising hour, 7.30 a.m. The pain was still present but had not lessened or increased and was not relieved by breakfast. About fifteen minutes after breakfast, while mixing a mash for the chickens, he developed once a period of five minutes a very severe pain in the same area. So severe and steady was this pain that he was forced to go to bed. He was very faint, covered with perspiration, cold and shivering as in a chill, very short of breath and thought he was going to die. After lying in bed doubled up with pain for an hour he vomited but got no relief. He was given a hypodermic which brought no relief. Belching of gas and more vomiting brought no cessation in the pain. Brandy was of no use and was vomited up. Two hours after the onset, he developed a sudden severe pain about the middle third of the left arm which radiated down to the fingers but did not remain there. An hour later (3 hours after the onset) he thought he obtained some relief from eating a piece of meat because the pain in the arm and epigastrium began to ease up and continued to do so. He was admitted to hospital late that night. On January 7th, 24 hours after the onset, there was only a slight pain in the epigastrium and a slight numbness in the arm. He felt weak and faint and was decidedly hungry.

Under previous illnesses patient gave the following: Two years ago about 7 a.m. he was suddenly seized with a pain in the left leg which radiated up to and down the left arm and over the left temporal region. He did not lose consciousness but was dazed for about a day. Partial paralysis of that arm and leg supervened and was followed by gradual improvement up to complete recovery in six months. There is no history of pneumonia, influenza or rheumatic fever. Five years ago he was in a street-car accident and was unconscious for a few hours, but otherwise, apparently, uninjured.

The family history was interesting in that his father died of a stroke at the age of 84 years.

Functional enquiry—No headaches or dizziness; no eye, ear, nose or throat trouble. He had a slight cough for the last two years with very little sputum.

There was some shortness of breath on moderate exercise but no palpitation. The appetite lately was good but from 6—25 years he had frequent bilious attacks with nausea and vomiting. The stools never showed any blood or were of an unusually dark colour. There was slight nycturia off and on for the last few years.

Physical examination of the cardiovascular system. The heart was enlarged to right and left as determined by percussion, measuring 5 cm. to the right in the 4th interspace and 10 cm. to the left in the 5th interspace. Except for an occasionol extra systole the heart rate was regular. The sounds could scarcely be heard even when he was turned on his left side. No murmurs were heard. There was marked varicosities of the veins of the legs, especially the right saphenous vein. The radial arteries were slightly thickened.

In the abdomen there was no rigidity, resistance or tenderness of the abdominal muscles. The liver was just palpable and slightly tender. The liver dullness was not decreased.

Slight pitting on pressure was observed around the ankles.

The patient was greatly worried and apprehensive about his condition.

On admission the temperature was 99° and the white blood count was 14,000. The pulse was 90.

The provisional diagnosis was:

1, Coronary thrombosis; 2, duodenal ulcer; 3, Chronic degenerative myocarditis with failure; 4, Generalized arteriosclerosis.

The patient remained in bed at the hospital and showed some improvement until January 16th, when he again took a severe pain in the epigastrium which radiated through to the back. He became increasingly cyanosed, dyspnoeic and restless, with a feeling of impending death. The pulse rate was regular but rapid. He vomited a number of times and was given 50% glucose intravenously, and morphine and atropine hypodermically. He died at 2.20 o'clock a.m., January 17th.

Comment—Here is an upper diaphragmatic condition resembling an acute abdomen. These resemblances may be so close at times that the primary cause of distress is sometimes not recognized. In this case the following points favoured coronary thrombosis as the primary cause of the distress:

1. The onset and nature of the pain, with its radiation to the arm and fingers.

2. The lack of rigidity, tenderness and diminution of liver dullness or abdominal examination.

3. The enlarged heart with weak and muffled sounds.

4. The absence of shock which usually accompanies perforation of duodenal ulcers.

5. The history of unilateral paralysis, undoubtedly of cerebral origin, two years previously.

Banti's Disease.*

By E. H. BENSLEY, B.A., 3T0, and J. E. GUNDY, 2T9.

This disease was first described in 1894 by Banti and, since that time, has borne his name. At that time he described the condition as a clinical disease entity, the chief manifestations of which were (1) splenomegaly, (2) secondary anaemia, (3) chronicity and (4) portal cirrhosis. As early as 1883 Banti had mentioned the occasional coincidence of portal cirrhosis and splenomegaly, but it was not until 1894 that he suggested that the condition was a disease entity and described it fully. In 1900 Osler studied the disease, drawing it to the attention of American physicians. He pointed out the frequent occurrence of hematemesis, adding this to the clinical manifestations described by Banti. In 1910 Banti published an account of the pathology of the disease. On the basis of clinical and pathological findings, Banti maintained that the splenomegaly was primary, regarding the disease as essentially one of spleen, and recommended splenectomy as a therapeutic measure. In the course of time it has been found that there are a number of conditions of known aetiology, in which the splenomegaly cannot be regarded as primary, and yet which may give rise to a clinical and pathological picture very similar to that described as characteristic of Banti's disease. The tendency has been to split off these conditions from Banti's disease, speak of them as cases of pseudo-Banti's disease and use the term Banti's disease only in cases in which the splenomegaly appears to be primary and no cause for the condition can be found. It is this conception of Banti's disease which has been adopted in this paper. However, some apply the term Banti's disease to some of these types of pseudo-Banti's disease and as a consequence considerable confusion in regard to the meaning of the term has arisen.

Pathology of Banti's Disease.

1. Gross Pathology of the Spleen. The spleen is markedly enlarged. It may weigh as much as one to three kilograms. It is engorged with blood. The cut surface has a grayish pink translucent appearance and sinks a little below the capsule due to contraction of the fibrous tissue which has become markedly increased in amount. The Malpighian corpuscles are not visible or, if visible, they are but few in number and widely separated. The fibrous trabeculae are thicker than normal. Haemorrhages, large infarcts and chronic perisplenitis with adhesions to surrounding structures are not infrequent.

2. Pathological Changes in the Blood Vessels of the Spleen.—The veins of the spleen are enormously distended, probably as a consequence of the increased blood flow through the spleen in this disease. Among the veins enlarged are the collateral vessels between the spleen and diaphragm and the vasa brevia which pass from the fundus of the stomach to near the hilus of the spleen to drain into the splenic vein. Normally the blood flows from the fundus of the stomach to the splenic vein in these vasa brevia. It is probable that, with an increase in the amount of blood which has to be carried by the splenic vein to the portal vein, some of the blood sidetracks the liver by passing through the vasa brevia to the fundus of the stomach and thence by veins in the stomach wall to the oesophageal veins at the cardia, which drain to the inferior vena cava, the flow in

*This article deals with the Pathology and Pathogenesis of Banti's Disease. Next month, as a sequel, the Symptomatology, Diagnosis and Treatment will be considered.

the vasa brevia thus being reversed. The hematemesis, which is so characteristic of Banti's disease, may be due to rupture of one of the distended branches of the vasa brevia in the wall of the stomach.

In a great many, but by no means all, cases there is an endophlebitis and calcification of the splenic vein, extending sometimes to the junction of the splenic and superior mesenteric veins and even into the portal vein itself. This is frequently accompanied by thrombosis in the splenic vein. If splenic or portal thrombosis occurs the blood from the spleen must leave the latter by collateral channels, namely the vasa brevia and the veins extending between the spleen and diaphragm, and if there is only splenic and no portal thrombosis, also by the left gastro-epiploic vein which forms anastomotic connections in the stomach wall with the right gastro-epiploic vein which drains into the superior mesenteric vein. It has already been pointed out that it is possible that the vasa brevia might become distended due to increased blood flow through the spleen. If splenic thrombosis occurs there will be still further distension of the vasa brevia and also distension of the gastro-epiploic veins increasing the likelihood of ruptures of a vein in the wall of the stomach, producing hematemesis. The thrombosis may involve the inferior mesenteric vein, a tributary of the splenic, producing congestion of the lower bowel which may lead to the formation of haemorrhoids and rupture of vessels with the appearance of red blood in the stools. If the thrombosis involves the portal vein ascites will develop. In regard to the relationship between splenic and portal thrombosis and Banti's disease it may be pointed out that such thrombosis may produce a picture similar to, if not identical with, Banti's disease. I refer in the preceding discussion, not to this, but to a thrombosis occurring in the course of a Banti's disease, the thrombosis being, not the underlying cause, but only an event occurring in the course of a disease which commenced before the thrombosis took place. However, some investigators believe that such thrombosis, presumed to occur late in the course of Banti's disease, never takes place, the thrombosis always preceding the pathological changes in the spleen and being the underlying cause. To determine, by post-mortem methods, the relationship in point of time of the thrombosis to the splenic changes is very difficult and the point, at present, is a controversial one. It is known that cases of Banti's disease, showing no thrombosis at operation, frequently develop thrombosis after splenectomy. Therefore, in considering the relation of thrombosis to Banti's disease, one may distinguish three possible types of thrombosis differing from one another in the time of their occurrence—(1) thrombosis which is the initial event and is the underlying cause of splenic changes which resemble those found in Banti's disease and which some state constitute true Banti's disease, (2) thrombosis occurring in the course of an untreated case of Banti's disease, in which the splenic changes precede the thrombosis, the latter probably being related to the endophlebitis of the splenic vein, and (3) thrombosis occurring after splenectomy has been performed in a case of Banti's disease which, at operation, showed no thrombosis.

3. Microscopic Pathology of the Spleen.—In the early stages of the disease there is hyperplasia of the Malpighian corpuscles and proliferation of the pulp cells. The latter show evidences of increased hemolytic activity in the form of engulfed red blood cells and deposits of hemosiderin. The spleen is engorged with blood, the sinuses being dilated. In these early stages the fibrosis is slight or absent.

In the later stages the most marked feature is the extensive fibrosis which has occurred. This fibrosis is described by Banti as commencing around the central artery of the Malpighian cor-

puscle (the intralobular artery) and spreading outward. As a consequence of this fibrosis many of the Malpighian corpuscles are destroyed and those that still remain are small. The fibrosis extends into the splenic pulp, many of the pulp cells being destroyed with the result that, in these later stages, the spleen has an empty appearance, being composed essentially of fibrous tissue in which the vessels are embedded.

4. Pathological Changes in Parts Other Than the Spleen.

(a) Liver—In the later stages of Banti's disease, when the changes in the spleen are advanced, portal cirrhosis of the lived develops giving rise to portal obstruction. This is characteristic of the disease and was described by Banti.

It is to be emphasized that this cirrhosis follows the splenic changes in Banti's disease. In primary portal cirrhosis, there is splenomegaly, fibrosis and atrophy of the Malpighian follicles —a picture similar to that seen in Banti's disease—but the splenic changes follow the cirrhosis.

(b) Bone Marrow—A moderate hyperplasia of the bone marrow is found in Banti's disease. However, the haematopoietic tissue of the bone marrow does not respond actively to the anaemia present in Banti's disease for examination of the blood shows little or no evidence of blood regeneration, reticulated cells and nucleated red cells being scarce or absent.

5. Disturbances in the Functions of the Spleen.—The activity of the spleen in destroying red blood cells is very greatly increased. This is shown (1) by the evidence of increased blood destruction seen on microscopic examination of the spleen in the form of a proliferation of the pulp cells and endothelial leucocytes with the presence in them of considerable numbers of engulfed red blood cells and deposits of hemosiderin and (2) by the increased excretion of urobilin in the urine. The latter is usually given as an indication of increased blood destruction in Banti's disease and probably this is correct but it may be pointed out that increased urobilin excretion may be produced by liver damage without increased destruction of red cells and that there is a certain amount of liver damage in Banti's disease. There does not appear to be any change in the influence of the spleen on the resistance of the red blood cells to hemolysis, their resistance being unchanged or only very slightly increased.

The influence of the spleen on blood platelets may be disturbed. Rosenthal has demonstrated that, in some cases of Banti's disease, the blood platelet count is normal or only slightly below normal but, in other cases, there is a definite thrombocytopoenia. These cases exhibiting a thrombocytopoenia show a marked haemorrhagic tendency. Possibly, in these cases, the activity of the spleen in destroying blood platelets is increased.

Summary of the Pathology of Banti's Disease.

1. *Gross Pathology of the Spleen.*
   - i splenomegaly
   - ii engorgement of the spleen with blood
   - iii Malphigian corpuscles scarce or absent.
   - iv fibrous trabeculae thickened
   - v often haemorrhage, infarcts, chronic perisplenitis with adhesions.

2. *Pathological Changes in the Blood Vessels.*
   - i veins draining spleen distended.
   - ii often endophlebitis and calcification of splenic and sometimes portal vein.
   - iii may be accompanied by splenic and sometimes portal thrombosis.

3. *Microscopic Pathology of the Spleen.*

   (a) Early stages.
   - i hyperplasia of Malpighian corpuscles
   - ii proliferation of pulp cells
   - iii many engulfed red blood cells and deposits of hemosiderin
   - iv blood sinuses dilated
   - v fibrosis slight or absent

   (b) Later stages
   - i extensive fibrosis and destruction of Malpighian corpuscles and pulp cells

4. *Pathological Changes in Parts Other than the Spleen.*
   - i. Liver—portal cirrhosis
   - ii Bone Marrow—moderate hyperplasia

5. *Disturbance in the Functions of the Spleen.*

i increased hemolytic activity of the spleen
ii possibly increased destruction of blood platelets by spleen in some cases.

Pathogenesis of Banti's Disease.

It may be stated at the outset that the pathogenesis of Banti's disease is not understood. The spleen appears to be the site of origin of the disease since, a cure has been effected by removal of this organ.

Suggestions regarding the pathogenesis of Banti's disease fall into three main groups:

(1) Those based on the results of attempts to demonstrate some infectious micro-organism, located in the spleen, as the cause of the disease. In the case of some investigations efforts were made to reproduce the disease in experimental animals. This method of approach has not proven fruitful. Mention should be made of the finding of a streptothrix in the spleen of certain cases diagnosed clinically as Banti's disease and the isolation of diphtheroid organisms from several spleens of cases of Banti's disease. Kristjanson has isolated a coccoid organism from a case of Banti's disease and claims to have reproduced a similar picture in the dog by intravenous injection. However, the majority of investigators, including Banti himself, have failed to isolate specific micro-organisms and it is not generally accepted that an infectious origin of the disease has been demonstrated.

(2) Suggestions regarding the pathogenesis based on a careful consideration of the pathology of Banti's disease. Pathological changes in the spleen are said to precede pathological changes occurring elsewhere. Banti laid emphasis on the fact that the fibrotic changes in the spleen antedate cirrhosis of the liver. Since the hepatic changes follow the splenic changes the injurious factor must act first upon the spleen and then upon the liver. It will be remembered that fibrosis commences in the Malpighian corpuscles around the intralobular artery and then spreads outward involving the splenic pulp. This has suggested that the injurious agent is brought to the spleen by the splenic artery. It has been found that, if india ink be injected into the splenic artery, the carbon particles first appear at the Malphigian corpuscles. If the injurious agent comes by way of the splenic artery it must also be distributed to all other parts of the body including the liver. In this case one must assume that the spleen is unusually sensitive to this injurious agent, if it be a toxin, or acts as a medium, more suitable than any other tissue, for the growth of the causative micro-organism, if such exists. It was suggested by Banti that some harmless substance is brought to the spleen by the splenic artery and there changed into a toxin which produces fibrosis in the spleen. This splenotoxin, as he calls it, is carried to the liver by the portal circulation and there produces a cirrhosis. It is generally assumed that the fibrotic changes in the liver are due to the same factor that produces fibrosis of the spleen. It seems to me that the liver damage might be related to congestion of the liver resulting from the increased blood flow through the spleen and the splenic changes due to some injurious agent acting only on the spleen.

Another aspect of the pathology of Banti's disease which has given rise to a suggestion regarding pathogenesis is the increased hemolytic activity of the spleen. It has been thought that the products of hemolysis might act as toxins producing the fibrosis. The factor which stimulates the spleen to increased hemolytic activity is unknown.

(3) Suggestions regarding pathogenesis made by those who refuse to consider that, after all conditions of known aetiology producing the picture of Banti's disease have been eliminated, anything is left. They settle the aetiology of Banti's disease by stating that

the conditions of known actiology described as resembling Banti's disease or as pseudo-Banti's disease are Banti's disease and they will not admit the existence of a group of unknown aetiology which most call true Banti's disease. Norris, Summer and Shapiro state that both the clinical signs and the pathological picture of Banti's disease may be given by syphilitic disease and so impressed are they by the correspondence between the pictures in these diseases that they express the opinion that Banti's disease is really a manifestation of visceral syphilis. Yet others report cases of Banti's disease in which there are no evidences of syphilitic infection. Warthin has maintained that an initial and invariable portal thrombosis is the underlying condition in Banti's disease. Undoubtedly, portal thrombosis may give rise to a picture almost, if not quite, identical with that described as characteristic of Banti's disease but many cases of Banti's disease have been reported in which portal thrombosis was not present.

It is evident that there are many conflicting views regarding the pathogenesis of Banti's disease, and these are based on very little actual information, as a consequence of which, in most cases, these views are not supported by their exponents with any degree of assurance. Not only the details, but the main facts of the pathogenesis, are yet to be learned.

The Earlier Diagnosis of Measles. is the title of a brief paper by Philip Moen Stimson in the New York Academy of Medicine Bulletin. It offers such a concise statement of our own teachings that we feel it is well to quote briefly from it. Of the four periods of the disease, those of incubation, of invasion, or catarrh, of the rash, and of convalescence, only the first two are discussed here:

In the last half of the period of incubation there may be a temporary loss of five or six ounces in weight and the blood count may show a lymphocytic leucopenia towards the end of the period.

The period of invasion or catarrh begins in the great majority of cases with fever. Within a half day or so, there next may occasionally be found a "measles line" or linear congestion in the mucous membrane of the lower eyelid, about a third of the way from the blepharal margin to the fornix. In ten children where a typical measles line of this sort in the eye was observed and the disease prognosticated at this stage, it subsequently became manifest in eight. The duration of the line as such is short, the injection spreading to become a general conjunctivitis when the other evidences of catarrh appear, such as coryza and cough, comprising with conjunctivitis the "three C's," characteristic of the second or third day of the disease and the cause of much of its spread to other people. The rash in the throat, or exanthem, usually precedes the "three C's," as may also a short-lived prodromal rash on the body. Koplik's spots inside the cheeks, and which have also been reported as having been found in the eyes, nose, and vagina, usually follow after the catarrh in order of appearance; hence measles patients have been contagious with their catarrh for an appreciable length of time before the finding of Koplik's spots establishes the diagnosis. The typical exanthem characteristic of the disease follows usually at least a day later, after the appearance of the pathognomonic Koplik's spots—often more.

Uncomplicated measles rarely kills, deaths being due almost without exception to the effects of secondary infections. Therefore every case of measles should be isolated at its onset from every other case. Experience in hospital and home has shown conclusively the

value of this measure, and the harm that follows not using it.

Therefore, this winter and every year, we must be prepared to make the earliest possible diagnosis of measles, to isolate the patient on the first suspicion, and to protect him from secondary infection.

Girolamo Legato. There lived in Florence a "Signor Legato" who devised a method for petrifying pathological preparations. He announced this discovery in Vieusseux's Anthology (1831) and, in his Florentine period, seems to have some 300 insects, fish, reptiles and other animals, with about 74 anatomical specimens, obtained from the hospital. From these 74 specimens he made a mosaic table of 214 cross sections which he obtained by sawing and polishing. After his death many attempts were made to get at the secret of his method, but without avail. In some of the specimens the process of petrifaction was not complete and no inferences could be drawn from the chemicals found in his laboratory after his death. What a loss to science, and what a golden opportunity this would have been for a modernistic foyer to the new pathology building.

The Annual Report of Dean Primrose on the activities of the Faculty of Medicine has recently been published and distributed. Graduates and undergraduates alike on studying this report have just cause to be proud of our medical school. The University of Toronto Medical School ranks high on this continent as a centre of medical education and research, and the results of the past year are of such a nature as to insure our maintaining this high esteem

This year, as in former years, many changes are noted in the personnel of the staff. Professor Macleod has returned to his native country, having occupied the Chair of Physiology with great distinction for the past ten years. Dr. William Goldie, Associate Professor of Medicine, has resigned. Dr. Roy Thomas has been appointed head of the Emergency Staff. Dr. F. A. Cleland, Assistant Professor of Obstetrics, has resigned, and Dr. F. W. Marlow, Associate Professor of Gynaecology, has been unable to take any active part owing to illness.

The following promotions and appointments have been made:

L. J. Irving, Associate Professor of Physiology.

D. T. Fraser, Associate Professor of Hygiene and Preventive Medicine.

J. D. Loudon, Assistant Professor of Medicine.

G. E. Wilson, Assistant Professor of Clinical Surgery.

R. R. Graham, Associate in Surgery and Clinical Surgery.

G. C. Cameron, Associate Professor of Medical Research.

All departments have been actively engaged in research work, and many excellent papers have been published during the past year. We shall endeavour to touch on some of these in the ensuing numbers of the Journal.

The Undergraduates have played their part in upholding Medicine as the leading faculty in supplying men to nearly all branches of Intercollegiate sport as well as turning out strong teams in all Interfaculty sports.

Thus the entire report is highly encouraging, and we would advise the Undergraduates to familiarize themselves with it in order to appreciate the privilege which is ours in attending such a school. Any who have not received copies of the report may do so by applying at the Medical Office.

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EDITORIAL.

Hospital Enlargement in Toronto.

During the past year there has been great expansion and improvement in hospital facilities in Toronto, and at the present time an ambitious program is being carried out. In this and subsequent issues of the Journal some of these improvements will be outlined.

The Toronto General Hospital last year by extending the wings of the main building, enlarged the public wards and added 105 beds. This brings the total for public beds from 540 to 645. When the new Clinical Medicine building is completed the present Pathology building will be at the disposal of the hospital and will be given over mainly to the departments of Urology and X-ray. According to the present plan the Department of Urology will maintain about 55 public beds and X-ray about 30, which will add this number to the total beds. There will be a great advantage in bringing patients under treatment by these depart ments into separate wards where they are readily accessible. By adding another floor the department of Out Patient Medicine will also be housed in this building.

The new Private Patients' Pavilion, which is now being constructed, is the product of much thought and planning, and as a result there will be many innovations which will make for efficiency and add to the comfort of the patients. As now being built, it is in the shape of a T and will have 320 beds, but in time another wing will be added, converting it into an H and bringing the total up to 540 beds. There will be

eight floors for patients, divided entirely into single rooms with the exception of eight double rooms. All rooms will have separate lavatories, and a direct telephone and buzzer to the main switchboard, by means of which nurses may be called. Among the innovations mentioned above is a telaudograph system, by means of which orders written at one station appear simultaneously at another station. As an example, a doctor writing a prescription will have it transmitted immediately to the dispensary, where it will appear in writing with his signature. The uppermost floor will be given over entirely to operating rooms, of which there will be 13, including delivery rooms. They will be equipped with every facility, and illuminated by the most modern methods. It is expected that the building will be ready for occupancy Jan. 1, 1930, and it will bring the capacity of the hospital up to 1,120 beds.

APPLICATIONS FOR ASSISTANT EDITORSHIP.

Applications will be received from the present fourth year up till April 1st, for the position of assistant editor for the year 1929-1930. Under the present policy of the Journal the assistant editor in the following year takes over the editorship, the year as assistant editor being in the nature of experience and training. Address applications to The Editor, Medical Journal, Room 107, Medical Building.

Faculty News.

By ART WALKER, 2T9.

The thirty-second annual renewal of Daffydil was held in Hart House Theatre on February 21 and 22, and concerning the performance, the generally expressed opinion was that it excelled Daffydils of recent years. All the skits were of high order and the committee in charge, under the able guidance of G. H. Hutton, in their preparations, omitted no details.

It has been no great secret that Daffydil, as staged of late years, has been warmly criticized by staff and students alike. It was felt by many that the calibre of the show had deteriorated and that unless the quality could be improved, Daffydil as an institution should be discontinued. In short, Daffydil this year was on probation, and unless a superior show were produced, the faculty were threatening to advise that it be discontinued.

Faced with this situation, the committee undertook the task of improvement. How well they succeeded is evidenced by the fact that there have been no criticisms and the great majority of those who attended felt the performance an excellent one. There were two innovations which contributed materially. First, the move from Convocation Hall to Hart House Theatre, where the setting is much more appropriate and the acoustic qualities infinitely better. In the second place, the services of a director were obtained. Mr. R. Stone's work in improving the dramatical features was obvious.

The Daffydil cup for the best skit was again won by 3T0, who now win it habitually. It was an operating-room scene set to music and was both origanal and humorous. Those who took part in it were M. J. Kelly, W. Young, C. C. Misener, J. McCullough, J. R. Rogers, R. J. Peer, J. W. Whaley, and Hugh Inksater. Two skits, that produced by the Freshmen year and that staged by second year, were tied for second place.

Epistaxis contained many original and interesting features and was a credit to its editor-artist, R. M. Mitchell.

Daffydil banquet was held on Satur-

day, Feb. 23, at the King Edward Hotel, and in every respect it was as previous Daffydil banquets have been.

* * * * *

Members of 2T9 attended their last class function on Saturday, March 2, when the Graduation Banquet was held at the Victorian room in the Prince George Hotel .

The toast to the University was proposed by H. R. Ziegler and responded to by Dr. E. S. Ryerson. O. L. Stanton proposed the toast to the Faculty, and Prof. Hunter, class adviser, replied.

One of the features of the evening was the presentation of the Athletic Stick to R. M. Mitchell by Dr. Easson Brown, honorary president of the Medical Athletic Society, and by Prof. W. B. Hendry. The Stick is annually presented to that member of the graduating year who is deemed to have done most to bring honor in Athletics to the faculty.

S. V. Railton, president of the sixth year, was toast-master.

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One of the most successful Junior Medical At-Homes was held in Hart House on Friday, Mar. 2. The affair was largely attended by members of the senior as well as the junior years. In many respects the party was the same as the Senior At-Home. Arrangements were made by a committee composed of the presidents of the lower years, H. D. Barner, J. Sinclair and C. E. Vaughan.

The Romance of Surgery.*

JOHN B. DEAVER, M.D., Philadelphia, Pa.

Man has always been, and, no doubt, always will be the paramount interest of mankind, whether it concerns his relation to his fellow-man, as portrayed by the historian and the novelist, or his relation to the unknown Being "whom he was blindly prompted from within to serve," or whether it concerns the mysteries of the human body as "the temple of the living God." It is therefore not only in the works of fiction and of the imagination that one need look for drama, but the thread of romance can be traced, now running bright, now less bright, throughout every aspect of the all-absorbing study of man. That the mysteries of the human body form no exception to this rule is readily seen in the fact that from time immemorial it has been the aim of the student and the investigator to fathom the hidden things, and even down to our own times the revelations they uncover partake of the spectacular, nay, almost of the unreal.

In the limited field of surgery this thread of romance can be traced step by step, especially in the unfolding of those formerly hidden lesions to which the human body is liable, and which have been gradually brought into view and to a large extent become amenable to alleviation and sometimes even a cure. Before proceeding, I wish to assure you that it is not my purpose to begin way back at the beginning, but to review some of the circumstances that have supplied the material for the later chapters in the romance of surgery.

An event which marks the early endeavors of the surgeon to relieve abdominal distress and which has a strong flavor of the romantic is McDowell's first operation for the removal of an ovarian tumor. Picture to yourself, if you can, a country doctor in the year 1809 traveling sixty miles on horse back on a winter's day to see a patient whose strength was being sapped by the rav-

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* Reprinted from the Nova Scotia Medical Bulletin in which it appeared as a reprint from the Journal of the Iowa State Medical Society.

ages of a pelvic tumor. The results of this visit are memorable alike for their far-reaching effect, and for the audacity of a surgeon and the courage of a woman. We next see this woman, on a bleak day in December, 1809, "with her pendulous abdomen resting on the pommel of her saddle," riding those same sixty miles, a two or three days' journey, into Danville, Kentucky, there to submit to the momentous experiment that was to supply the foundation for modern abdominal surgery. The central figures in these two pictures are Ephraim McDowell, the fearless surgeon, and Jane Todd Crawford, the heroic woman. Their story is indeed worthy of the pen of the novelist.

The description of the operation as it has come down to us from the pen of McDowell himself is not without interest both for its extreme simplicity and the apparent unconsciousness of the surgeon of the import of the procedure.

With the patient on a table on her back, an incision was made three inches from the left rectus abdominis muscle and extending for nine inches, parallel with the fibers of the muscle, into the abdominal cavity. The tumor was then exposed and was so large that it could not be removed in its entirety. A strong ligature was put around the Fallopian tube near the uterus, and the tumor then cut open. It was found to consist of the ovary and the fimbriated part of the tube, very much enlarged. Fifteen pounds of a dirty gelatinous-looking substance were removed, the Fallopian tube was then cut through and the sac extracted, the weight of which was seven and one-half pounds. As the incision was made the intestines popped out upon the table and the abdomen was so completely filled by the tumor that the intestines could not be replaced during the operation, which lasted about twenty-five minutes. The patient was then turned upon her left side to allow the blood to escape, after which the incision was closed with interrupted sutures, the end of the ligature around the Fallopian tube being allowed to protrude from the lower end of the incision. Between every two stitches a strip of adhesive plaster was applied, which "by keeping the parts in contact, hastened the healing of the incision." The usual dressings were applied and the patient put to bed, "a strict observance of the antiphlogistic regimen" being prescribed. When McDowell visited his patient five days later he found her engaged in making her bed. He gave particular directions as to the future care of her health, and twenty-five days after the operation she was again seen seated on horseback travelling those same sixty miles back to her home, where she remained in good health until her death, thirty-five years after the epochal event in surgery in which she had so valiantly figured.

McDowell subsequently performed two similar operations but did not report them until several years later. In fact, the criticism aroused by the procedure may have prevented an earlier publication of the method. The abdomen was always considered as too sacred for the knife and the female generative organs as the most sacred part of the abdomen. So much so in fact, that in this country the operation was not again heard of until 1821, and abroad the first of its kind is reported from Germany in 1819, while in England it had not been attempted until 1836, and in France, until 1844.

The successors of McDowell, Dunlap, Peaslee, Kimball and the two Atlees, worked hard against the most adverse criticism to remove the prejudice that existed against the operation. Considering the boon that this one procedure has been to suffering mankind, does it not seem as though these intrepid pioneers had reversed the proverb and become angels who rushed in where others were afraid to tread?

For the next episode in our romance the scene changes from the Kentucky backwoods to the more cultured atmosphere of the city of Boston, when "hope mid deepening stillness watched one eager brain, with God-like will decree

the death of pain," as on October 16, 1846, in the operating room of the Massachusetts General Hospital, there took place the first public demonstration of the use of ether anesthesia in operative surgery. The central figures of this dramatic event were W. T. G. Morton, a dentist, the anesthetist of the occasion, John C. Warren, the surgeon, and a young man, whose name has not come down to us, willing to submit to the experiment of painless surgery for the removal of a tumor of the neck, just below the mandible. The incision was made and the tumor removed, the patient after returning to consciousness declaring he had felt no pain, although he was aware of the operation, stating that "the stroke of the knife felt like a blunt instrument being passed roughly around his neck."

Contending claims of priority in the use of ether anesthesia have established the fact that Crawford W. Long of Jefferson, Georgia, had antedated Morton by four years, and to him belongs the credit of priority in its use. On March 30, 1842, Long administered ether to one James M. Venable, "till completely anesthetized and then excised a small cystic tumor from the back of his neck." The surprise of the patient on regaining consciousness can well be imagined as he saw the tumor in the hand of the surgeon, while he had not felt as much as a scratch.

It is not surprising that in 1846 Morton in Boston should not have known of Long's discovery in Georgia. Distances at that time were much greater than at present, and the habit of rushing, or being unwillingly rushed into print, had not yet become established. Nor is it remarkable that two surgeons should simultaneously and quite independently have come upon the same idea. The horrors of the operating room were such that the desire to reduce pain was ever uppermost in the minds of the operators of those days. Indeed, the idea of an anesthetic had long been conceived, judging by the crude attempts of a former day. It is, however, the final birth of this priceless boon which has the flavor of fiction. It appears that even in those pre-Volstead days a fashionable form of amusement was to produce intoxication by nonalcoholic means. These sprees took the form of nitrous-oxide parties, at which the participants would become gloriously drunk from inhaling laughing gas provided by itinerant lecturers on chemistry after the close of the lecture or entertainment. The fashion spread even to the obscure town of Jefferson, Georgia. On one occasion a group of students begged Dr. Long to give a nitrous-oxide party. In the absence of the material for preparing the gas, Long suggested that sulphuric ether would serve the same purpose. The party proved a great success, and after that, ether frolics became the rage. Long and his friends, the morning after such parties, often found bruises on their bodies without any knowledge of how they had been received. It was then the idea came to Long that "ether must have the power of rendering one insensible to pain, and should therefore be available for preventing pain in surgical operations." The fashion of nitrous-oxide exhilaration spread even to staid New England. We find that Horace Wells, a dentist of Hartford, Connecticut, discovered the anesthetic powers of nitrous-oxide in much the same way. After an entertainment given by a popular lecturer on chemistry, he saw a man under the intoxication of laughing gas injure his ankle without experiencing any pain, and at once concluded that nitrous-oxide must be powerful anesthetic. He proved his case by using it for extracting a tooth of a patient, and afterwards continued to use the gas in his profession of dentistry. Associated with Wells at the time was one Jackson, and it was through the latter that Morton conceived his idea of using ether as an anesthetic. At that time ether was a well-known antidote to relieve the pain and dyspnea following the use of chlorine gas poisoning. Jackson had often used it in

that way, but it was left to Morton to demonstrate its efficiency in surgery. Thus from the idle frolics of youth the dream of the surgeon was suddenly realized and assumed practical form and shape.

The domain of surgery was now suddenly widened, but the obstacles to successful surgery were by no means as suddenly removed. For with the rapid increase of the scope of operations that followed the introduction of anaesthesia, the grim spectre known as hospital gangrene loomed larger than ever before. To rout this ogre from his lair and lay him forever low now became the ambition of the surgeon.

Across the waters in a small village in the mountains of eastern France a youth was wavering between art and science in the choice of a career. Fortunately for mankind science won the day, and to a chemist who had never handled a scalpel and who had to screw up his courage to witness a surgical operation, who did not even know that it was possible to bore a hole in the skull of animals for experimental purposes and in man for therapeutic purposes, to this man the medical profession owe a debt which they freely acknowledge when they hail Pasteur as their leader "who led them into the kingdom which they longed for but could not have found for themselves."

From his native village of Arbois, Pasteur soon wandered to Paris, where he and his cohorts fought night and day to bring the light of the truth to the doubting and stubborn profession of the day—truths which had been revealed to Pasteur in almost as strange, though less frivolous a manner, as attended the birth of ether anesthesia. It certainly seems a long way from the study of the diseases of wines and of silkworms to the diseases of man. But, as you all know, such were the beginnings of Pasteur's epochal and romantic revelations, whose influence on the welfare of mankind transcends all imagination. Like pioneers of all times, Pasteur was compelled to fight a fierce battle for his convictions. While he was valiantly striving to maintain his position by brilliant and fearless demonstrations of the cause of wound infection, interrupting the discourses of learned doctors, as he sprang from his seat and drew on a blackboard the shapes of the microscopic organisms that were the offenders in the mysteries of inflammation, his theories were being quietly absorbed and were receiving their rightful recognition across the channel in the seclusion of a physiological laboratory and a surgical operating theatre.

The story of how Lister supplemented and applied Pasteur's discoveries is perhaps the most fascinating part of our romance. Lister's appointment to the chair of surgery in the University of Glasgow and surgeon to the Glasgow Royal Infirmary, in the year 1861, came to him after he had made important experimental studies on the early stages of inflammation, coagulation of the blood, etc. These studies led him to correlate sepis with putrefaction and to the realization that if putrefaction could be prevented sepsis could be avoided. But in the absence of a known cause for putrefaction he could think only of cleanliness as a means of controlling the putrid products of operative and other wounds. His efforts, of course, were futile. Hygienic cleanliness was still unborn.

It is easy to imagine that one of the chief topics of conversation among surgeons of that day was this ever-present spectre of infected wounds. It appears that on one of these occasions as Lister was walking home with one of his colleagues, Thomas Andrews, the professor of chemistry in Glasgow, as usual the subject of putrefaction came up. Thomas called Lister's attention to certain publications by Pasteur on the controversial question of spontaneous generation, the cause of fermentation, etc. Lister at once procured these papers. And lo, and behold, the solution of the riddle of the ages seemed at hand. The enemy resided not in the

gases of the air as surgeons had hitherto surmised, but in minute living organisms, floating in the air and present in the dust that settled on surrounding objects. And now one of Pasteur's favorite aphorisms, "Chance favors the prepared mind," was to find its most dramatic and far-reaching application. Lister's was a philosophical, logical, and experimentally inclined brain. His general conclusions upon the revelations of Pasteur's work were: Prevent the germs from entering the wound—and secondly, if once they have entered, kill them before they have a chance to do further mischief, or, as proved by later developments, inhibit their growth without the necessity of killing them.

And thus the campaign for the battle royal against infection was set, and as we know, culminated in the most signal victory in the warfare of science and in the annals of mankind. The first event in the battle took place on August 12, 1865, in the Glasgow Royal Infirmary, the principal actors on that occasion being Lister, his house surgeon, MacFee, and James Greenlees, a lad eleven years of age, who had been admitted with a compound fracture of the left leg, which Lister treated by his new antiseptic method. To Lister the most remarkable fact in the result was the immediate conversion of the compound fracture into a simple fracture with a superficial sore.

The surgical world was soon divided into Listerians and non-Listerians. The controversy raged for a decade or more, but gradually subsided until none were left to decry this inestimable boon to suffering humanity. The Revolution of Surgery is the fitting heading to this chapter in the Romance of Surgery, for it has entirely changed our manner of thinking and of working both in the home and in the hospital. A very interesting part of our tale could be written on the hospital then and now, but time does not permit the digression.

The outstanding effects of the revolution are the throwing down of the barriers in the field of surgery together with the reduction of operative mortality; in other words, the comparative safety of surgery. For there is no doubt that to-day we operate with a certainty of results which could scarcely have been dreamed of in a former day.

From the head downwards there is scarcely any part of the body that has not been successfully subjected to surgery. Even the heart is no exception and the abdomen and the pelvis, formerly considered as sacred precincts, have almost become the "playground" of the surgeon.

The invisible lesions, especially those of the abdomen, could now be exposed. One of the first fallacies to be expelled by this freedom was the idea of idiopathic peritonitis. Peritonitis, arising from within the abdomen, was soon recognized as a lesion secondary to a primary source of inflammation.

To our ancestors peritonitis was a familiar term. And if the head-stones in our grave-yards could relate more than the usual "hic jacet," we could easily compile statistics of the toll taken by the grim reaper from this one condition alone.

The recognition of the appendix as one of the most potent primary factors in peritonitis forms an interesting chapter in the tale we are trying to unfold.

There was little of the romantic in the controversy that arose around this question of primary peritonitis versus appendicitis or some other visceralitis —if I may be allowed to coin the term. But what seems almost as strange as both fiction and truth often appear to be, is that the many astute observers of a former day should have been so close to solving the problem of phlegmon in the region of the right iliac fossa without having come upon its solution. No less strange is it that in the face of the overwhelming force of facts, which neither theory could dispel nor speculation controvert, the profession was so slow to concede the rationale of appendectomy, after once the surgeon had

gained the courage to enter the peritoneal cavity.

The opposition came from some of the most distinguished members of the profession. On one occasion (May 2, 1892), in discussing a paper read by me before the College of Physicians of Philidelphia, the late Dr. J. M. DaCosta, while agreeing with me on certain points, stated that he was not prepared to go so far with me as to advocate operation for every case of appendicitis, even in the first attack. To him it seemed that a person with appendicitis would go to bed, not with the sword of Damocles suspended over his head, but with the scalpel of the surgeon already on his abdominal wall. The reverse has come to be true, for in most instances unless the scalpel of the surgeon is at once on the patient's abdomen, the sword of Damocles is actually suspended over his head in the shape of recurrent attacks, and possible perforation and perhaps death. The battle is not yet entirely won and will not be until the nine-lived procrastinator finally disappears. Not until then will surgery enjoy the complete triumph of having added at least one to the list of curable diseases.

Because of the hidden seclusion of the appendix, it failed of recognition for so long as the malefactor in so many cases of abdominal inflammation. The rôle of other organs as the seat of disease was better known, but treatment was necessarily only systemic until it was found by animal experimentation, especially directed toward a better knowledge of the physiology of the human body, that the removal of certain viscera was entirely compatible with life.

The thread of romance at this point is the interesting and fascinating demonstration resulting from the various "ectomies" that are being practised today, of the law of compensation provided by a beneficent nature. The removal of one of a paired organ like the kidneys was the first to be attempted—and we know that the remaining kidney is capable of performing the function of both. Later the removal of single organs, such as the gall bladder and the spleen, was attempted. In the absence of the gall-bladder the cystic duct takes on part of the function of the missing organ; regeneration of splenic tissue is a well-known fact. This remarkable provision is so taken for granted to-day that we often forget to stop and marvel —"Can such things be, and overcome us like a summer cloud without our special wonder?"

Within the gastrointestinal tract it is the possibility of resection and anastomosis that should excite our special wonder. While most of these operations were deliberately devised and tried out by animal experiment, there is one which we owe to sudden inspiration and which now (with certain modifications) is an every-day occurrence in every large operating clinic. Not only that, but it has become as fruitful a source of controversy as pertained to the early days of appendectomy.

It is no doubt a familiar story to you how gastrojejunostomy originated —the scene is laid in Billroth's clinic in Vienna in the year 1881. The participants are Wolfler, the surgeon, Nicoladini, his assistant, and the patient—a man suffering from pyloric obstruction. Standing at the side of Wolfler in the presence of what proved to be an inoperable carcinoma of the pylorus—Nicoladini, as Wolfler was about to close the abdomen, suggested that a new passageway for the food could be provided by anastomosing the small bowel to the anterior wall of the stomach. It was a bold suggestion, especially in that the anastomosis was made in the anti-peristaltic direction, but fortunatly the operation proved a success. The patient made a good operative recovery and four weeks after the operation was discharged in good condition. The defects of the procedure became apparent in the second operation performed by Wolfler. The patient died and at autopsy it was found that a vicious circle had formed as the result of

spur-formation. The publication of Wolfler's experience aroused the interest of the surgical world. Increasing observations and experience gradually led to changes and modifications in the procedure, as we know them to-day. As I have already said, the subject of gastrojejunostomy provided, and still provides, a very fruitful source of discussion to this day, for it soon became apparent that the method would be of value also in the treatment of benign diseases of the stomach and duodenum, especially ulcer, which, like the poor, we have with us always.

At the outset I promised not to take you back to the beginnings of our romance; by this time, I am sure you are hoping that I won't keep you long enough to bring the story up to date. Indeed, it would be a stupendous task, for almost every day we are brought face to face with something new to excite our wonder that such things can be.

If I may be permitted a glance into the future, I would say that the immediate trend seems to be toward more surgery, which by actual sight is unfolding pathology as it exists in the living subject. In a more remote time it may be that the correlation of the pathologic facts thus revealed with the end results of operations in large numbers of cases, together with the further development of the chemistry of the human organism, may lead to the day when less surgery will be required for internal diseases. Be that as it may, the present era will doubtless be known as the surgical era, to which future generations will acknowledge their indebtedness in equal measure with their obligation to Pasteur and Lister.

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